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Dr Andrew Padula is a veterinarian with over 25 years of experience in veterinary research, industry and clinical practice, and the founder of Padula Serums. The company is an APVMA licensed biologicals manufacturer of animal derived antisera for therapeutic and research purposes. Dr Padula has had a long fascination with venomous creatures and the application of the life sciences to treat their potentially lethal effects.

Dr Padula obtained a PhD from the University of Melbourne and is currently an honorary senior research fellow within the Australian Venom Research Unit at the University of Melbourne. He holds a European veterinary specialist qualification in production animal reproduction and medicine.

Dr Padula is a former veterinary practice owner in Gippsland for over 10 years and has published numerous papers on snakebite in domestic animals and tick paralysis.

To read more about RBBS envenomation and treatment in companion animals check out the snakebite information page on the Padula Serums website (www. padulaserums.com.au/info/snakebite).

Red-bellied black snake envenomation in a dog requiring blood transfusion

This case in a puppy demonstrates that red-bellied black snake (RBBS) venom has the potential to cause severe and delayed life-threatening haemolysis requiring blood transfusion. The RBBS (pictured below) is one of the most common causes of snakebite in dogs and cats in eastern Australia. Although no human deaths are recorded in Australia in modern times from this snake, it is potentially lethal for dogs. Envenomation by this snake species can cause unexpected problems in clinical management with potential for prolonged and ultimately fatal outcome. In the author's experience approximately 10% of RBBS cases in dogs will require blood transfusion.

A 14-week-old 15kg entire female German shepherd dog came for veterinary treatment after-hours due to vomiting, salivation and general lethargy. Earlier in the day the owner described the dog as entirely normal. The owner had historically observed RBBS on their Gippsland property and snakes were known to be active at this time of year.

On initial examination the dog was mildly distressed and was making repeated attempts to swallow. Both front legs were covered with saliva because the dog had been hypersalivating extensively. A soft, nonpainful swelling, approximately 4cm diameter was present under the jaw on the ventral aspect of the neck. Based on the clinical signs and history, a RBBS envenomation was suspected. Oedematous swellings over puncture sites are commonly seen with RBBS and in some cases the swelling can be very extensive. Snakebite from other members of the black snake family (Pseudechis sp.) also cause soft tissue swelling, and in human cases, finger or even arm amputation has resulted. In the author's experience RBBS will occasionally cause severe tissue destruction, particularly with bites around the ears.

The dog was hospitalised and placed on intravenous fluids (0.9% NaCl). An activated clotting test was performed using whole blood and was mildly prolonged (100 s). Both the prothrombin time (18.0 s) and activated partial thromboplastin time (84 s) were also mildly prolonged. The PCV at presentation was 43%. Haemoglobinuria was noted in a urine sample collected at this time. A plasma sample collected at the same time had only minor gross haemolysis. RBBS venom causes only minor blood coagulation disturbances compared to tiger and brown snake venom which have very active procoagulant toxins in their venom.

The dog was treated with one vial of tiger-brown snake antivenom 8000 units



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Clockwise from right: On

FOR ANIMAL TREATMENT ONLY Tiger-Brown Snake Antivenom 8000 units

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presentation, a soft, non-painful swelling, about 4cm in diameter, was observed under the dog's jaw; initial treatment was with one vial of tigerbrown snake antivenom; in a urine sample haemoglobinuria was noted; the geographical distribution of the red-bellied black snake.



(Padula Serums Pty Ltd, Australia) by slow bolus intravenous infusion. The antivenom is licensed with the APVMA and has registered claims against tiger, brown and black snakes. One hour after antivenom infusion the dog was noticeably brighter and seemed less anxious. Tiger snake antivenom is recommended in human cases of RBBS instead of the more specific black snake antivenom which is larger volume and higher cost. There are common components between tiger and RBBS venom that are effectively neutralised by tiger snake antivenom and also explain why RBBS venom reacts in both tiger and black snake wells in a snake venom detection kit.

The next day, 14 hours after antivenom administration, the dog appeared clinically normal, eating well and was sent home with the owner. However 36 hours later the dog returned because it was again lethargic. A full clinical haematology and biochemistry profile at this time revealed a PCV of 19% (37-55) with haemoglobin 54 g/L (115-180) and evidence of regenerative anaemia with 6.9% reticulocytes. The blood smear contained morphological abnormalities of the red blood cells including rouleaux formation, polychromasia and a mild thrombocyotopaenia.

Red cell morphological changes are likely due to the phospholipase activity of the RBBS venom damaging the sensitive lipid bilayer resulting in a swollen and rounded appearance. Spherocytes are typically associated with immune mediated haemolytic anaemia conditions, but the mechanism appears different following RBBS envenomation. Dog RBCs seem particularly sensitive to the haemolytic toxins and their presence is almost pathognomonic in dogs for RBBS envenomation.

Serum biochemistry at this time revealed CK 1363 U (<400 U), ALT 329 U/L (<80) and ALP 164 U/L (<120 U/L). The dog was hospitalised and given a transfusion of 450 mL of freshly collected whole blood. The dog's condition then improved markedly and after a further 24 hours of hospitalisation it was discharged without further problems.

Humans envenomed by RBBS often describe a very painful abdomen associated with vomiting and nausea, which following antivenom administration rapidly subsides. This canine case appeared to benefit immediately from antivenom with visibly reduced anxiety, which could have been abdominal pain related. Loss of taste and smell, or a constant metallic taste in the mouth for over one year has occasionally been reported in humans following RBBS envenomation.

In human cases, antivenom is often withheld unless there is evidence of severe envenoming. The same cannot be said of dogs in which the author has observed rapid death (<1 hour), respiratory paralysis, extensive soft tissue destruction and death from renal failure. Unfortunately, many owners consider this snake to be less venomous than what it is and may delay presentation of dogs until very unwell, often with acute kidney injury and renal failure developing.

Retrospective measurement of the RBBS venom concentration by a specific snake venom research ELISA demonstrated a high level of venom in serum at initial presentation (52 ng/mL) and urine (1,300 ng/mL). No venom was detectable in serum after antivenom administration. These venom concentration values are typical for RBBS cases studied and also show that urine is a good matrix for running the commercially available snake venom detection kit on due to higher venom concentration. The absence of venom after antivenom indicates free venom had been effectively bound to antivenom.

This case highlights the potential for complications arising from ongoing haemolysis caused by RBBS venom components. Monitoring of PCV and RBC morphology is useful to follow recovery and indicate if a blood transfusion may be required. Acute kidney injury leading to renal failure is also a potential complication due to dehydration, haemolysis and possibly direct effects of the venom.

Veterinarians should not underestimate the potential lethality of the Australian RBBS and be prepared to treat assertively with antivenom and monitor blood parameters to minimise potentially fatal complications. **V**